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1: Eur J Neurosci. 1996 Jun;8(6):1149-55.

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Activation of nucleotide receptors inhibits high-threshold calcium currents in NG108-15 neuronal hybrid cells.

Filippov AK, Brown DA.

Department of Pharmacology, University College London, Gower Street, London WC1E 6BT, UK.

A P2U (UTP-sensitive) nucleotide receptor has previously been cloned from NG108-15 neuroblastoma x glioma hybrid cells and it has been shown that activation of this receptor inhibits the M-type K+-current. We now report that UTP also inhibits Ca2+-currents in differentiated NG108-15 cells, but probably through a different nucleotide receptor. UTP (100 microM) inhibited the peak of the high-threshold current by 28.4 +/- 3.1% (n = 28) with no effect on the low-threshold current. Two components of high-threshold current were identified: one inhibited by 100 nM omega-conotoxin (CgTx) and one inhibited by 2 microM nifedipine and enhanced by 1 microM BAY K8644. UTP inhibited the former by 31.0 +/- 3.1%, with an IC50 of 2. 8 +/- 1.1 microM, and the latter 34.2 +/- 6.1% with an IC50 of 1.7 +/- 1.3 microM. Pertussis toxin pretreatment prevented inhibition of the CgTx-sensitive, nifedipine-resistant but not CgTx-resistant current. Inhibition was not prevented by intracellular BAPTA (20 mM) or cAMP (1mM). Effects of UTP on both currents were imitated by UDP, ATP, ADP, AP4A and ATPgammaS but weakly or not at all by 2-MeSATP, GTP, AMP-CPP or ITP. Since the receptors which inhibit Ca2+-currents are activated by ATP, it is suggested that they might mediate auto-inhibition of transmitter release by ATP if present on purinergic nerve terminals.
Keywords: nucleotides, UTP, ATP, calcium currents, neuroblastoma cells

PMID: 8752584 [PubMed - indexed for MEDLINE]

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